Single photon emission computed tomography evaluation for severe neuropsychiatric systemic lupus erythematosus

Sirs,

Neuropsychiatric symptoms are among the most significant and dangerous complications for SLE patients. MRI scanning has been the most common clinical diagnostic technique for NPSLE but it has limited diagnostic usefulness (1). More sensitive techniques to promptly diagnose non-typical and early-stage NPSLE are called for.

Original work: Fourteen severe NPSLE subjects with normal MRI findings, 18 SLE subjects with no NP involvement and 20 healthy subjects underwent ^{99m}Tc-ECD single photon emission computed tomography (SPECT). The NPSLE and SLE patients who were obtained from the Department of Rheumatology at the Xiangya Hospital fulfilled ≥4 of the 1997 American College of Rheumatology (ACR) revised criteria for SLE (2). All severe NPSLE patients (13 women and 1 man) manifested neuropsychiatric syndromes that included epilepsy (n=7), psychosis (n=3), depression (n=2), cranial neuropathy (n=1), and, Guillain-Barré syndrome (n=1). Severe NPSLE subjects' mean age±SD was 30.5±9.8 years with illness duration 7.6±5.4 years. Disease activity index (SLEDAI) was prepared for each patient. A follow-up SPECT was performed on all subjects two months after treatment. The aim of this study was to evaluate SPECT for diagnosing and treating severe NPSLE. We expected the use of SPECT brain imaging to monitor therapy in severe NPSLE.

Research findings: SPECT detected abnormalities in all (100.0%) of the severe NPSLE subjects having normal MRI findings thus demonstrating SPECT utility. The current understanding (3) that coupling MRI with SPECT improves clinical diagnostic accuracy was confirmed. Moderate to severe hypoperfusion involving multiple regions, particularly the temporal and frontal lobes, was the most common SPECT finding. Severe NPSLE subjects' clinical symptoms included seizures, psychosis and depression indicating likely temporal and frontal lobe involvement (4). The study combined semi-quantitative methods with cerebral blood flow changes in severe NPSLE subjects to evaluate SPECT utility and found that NPSLE subjects had significantly lower temporal and frontal region semi-quantitative values compared to SLE and healthy subjects (Table I). We conclude that NPSLE pathogenesis is vascular pathological change resulting in neuropsychological change and that severe NPSLE is more likely to involve the middle cerebral artery and its region. Further research into combining various diagnosis methods (5) to improve the clinical diagnostic accuracy is called for. Intravenous drip methylprednisolone (MP) followed by lumbar puncture intrathecal slow injections of methotrexate (MTX) plus dexamethasone(DXM) was administered to fourteen severe NPSLE subjects twice a week. CNS symptoms in all severe NPSLE improved and their SLEDAI score decreased significantly indicating effective treatment. SPECT imaging showed perfusion defects significantly improved, or even disappeared, in severe NPSLE subjects. Increased semi-quantitative values indicate temporal and left frontal cortex recovery (Table I). SPECT imaging results were consistent with the effect of treatment. Regional cerebral blood flow (rCBF) was restored by effective treatment. Future studies are needed to confirm whether severe NPSLE can be treated by intervention to change rCBF and vascular permeability.

This study indicated that SPECT represents a sensitive tool to detect the severe NPSLE. By semi-quantitative analysis, SPECT can objectively detect haemodynamic changes that is useful in follow-up, particularly for guiding treatment.

There are some limits in the current study. Replicating the study in a larger patient population is called for.

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References

1. KOZORA E, WEST SG, KOTZIN BL, JULIAN L, PORTER S, BIGLER E: Magnetic resonance imaging abnormalities and cognitive deficits in systemic lupus erythematosus patients without overt central nervous system disease. *Arthritis Rheum* 1998; 41: 41-7.
2. HOCHBERG MC: Updating the American College of Rheumatology revised criteria for the classification of systemic lupus erythematosus. *Arthritis Rheum* 1997; 40: 1725.
3. CASTELLINO G, PADOVAN M, BORTOLUZZI A *et al*.: Single photon emission computed tomography and magnetic resonance imaging evaluation in SLE patients with and without neuropsychiatric involvement. *Rheumatology* 2007; 47: 319-23.
4. GIOVACCHINI G, MOSCA M, MANCA G *et al*.: Cerebral blood flow in depressed patients with systemic lupus erythematosus. *J Rheumatol* 2010; 37: 1844-51.
5. NOWICKA-SAUER K, CZUSZYNSKA Z, SMOLENSKA Z, SIEBERT J: Neuropsychological assessment in systemic lupus erythematosus patients: clinical usefulness of first-choice diagnostic tests in detecting cognitive impairment and preliminary diagnosis of neuropsychiatric lupus. *Clin Exp Rheumatol* 2011; 29: 299-306.

Table I. Semi-quantitative values and SLEDAI scores comparisons in NPSLE and controls between pre- and post-treatment.

Region	NPSLE			SLE			Healthy		
	pre-	post-	*p*	pre-	post-	*p*	pre-	post-	*p*
L frontal	0.82 ± 0.05[#]	0.93 ± 0.04	0.032	0.90 ± 0.08	0.91 ± 0.02	1.231	0.93 ± 0.03	0.92 ± 0.04	1.427
R frontal	0.83 ± 0.07[*]	0.88 ± 0.05	0.569	0.91 ± 0.02	0.92 ± 0.05	1.450	0.94 ± 0.04	0.91 ± 0.02	2.112
L tempora	0.79 ± 0.02[△]	0.89 ± 0.03	0.003	0.90 ± 0.04	0.91 ± 0.02	1.325	0.95 ± 0.05	0.95 ± 0.07	3.215
R temporal	0.75 ± 0.06[▲]	0.87 ± 0.05	0.004	0.90 ± 0.07	0.92 ± 0.03	1.234	0.95 ± 0.06	0.94 ± 0.03	2.130
L parietal	0.90 ± 0.05	0.91 ± 0.02	2.149	0.91 ± 0.09	0.92 ± 0.03	2.147	0.94 ± 0.05	0.95 ± 0.03	1.657
R parietal	0.92 ± 0.05	0.93 ± 0.03	1.577	0.94 ± 0.05	0.94 ± 0.02	2.158	0.93 ± 0.02	0.94 ± 0.02	1.659
L occipital	0.92 ± 0.05	0.94 ± 0.06	1.781	0.92 ± 0.03	0.93 ± 0.02	1.384	0.93 ± 0.05	0.93 ± 0.02	1.264
R occipital	0.90 ± 0.03	0.90 ± 0.07	1.942	0.89 ± 0.03	0.91 ± 0.06	2.012	0.94 ± 0.04	0.92 ± 0.05	1.035
L basal ganglia	0.90 ± 0.06	0.90 ± 0.06	0.123	0.90 ± 0.06	0.90 ± 0.06	0.188	0.96 ± 0.06	0.95 ± 0.06	0.132
R basal ganglia	0.88 ± 0.09	0.90 ± 0.02	2.118	0.91 ± 0.04	0.92 ± 0.02	0.950	0.91 ± 0.02	0.93 ± 0.04	0.952
L thalamus	0.91 ± 0.04	0.92 ± 0.03	0.325	0.91 ± 0.04	0.94 ± 0.04	0.687	0.96 ± 0.03	0.95 ± 0.02	0.356
R thalamus	0.90 ± 0.05	0.94 ± 0.02	0.641	0.90 ± 0.07	0.92 ± 0.03	0.458	0.97 ± 0.04	0.98 ± 0.02	0.214
L white matter	0.90 ± 0.07	0.91 ± 0.02	0.588	0.91 ± 0.03	0.90 ± 0.05	0.231	0.97 ± 0.05	0.96 ± 0.04	0.135
R white matter	0.90 ± 0.05	0.92 ± 0.04	0.781	0.92 ± 0.03	0.93 ± 0.02	0.342	0.96 ± 0.06	0.95 ± 0.03	0.642
SLEDAI	23.8 ± 5.6	8.9 ± 5.6	0.004	6.6 ± 5.8	4.4 ± 3.6	1.237	NA	NA	NA

L: left; R: right. Comparing pre-treatment semi-quantitative values from the bilateral frontal, and bilateral temporal lobes between Severe NPSLE subjects and control subjects, there were statistically significant (p<0.01). [#]p=0.005,[*]p=0.004;[△]p=0.003;[▲]p=0.001.